Opinion

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A Brief Note on Neuropharmacology

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Description

The study of how medications alter cellular function in the nervous system, as well as the neurological mechanisms by which they influence behaviour is known as neuropharmacology.

Neuropharmacology is divided into two branches: Behavioral and molecular. The study of how drugs affect human behaviour (neuro psychopharmacology) is a focus of behavioral neuropharmacology, as is the research of how drug dependency and addiction affect the human brain. The study of neurons and their neurochemical interactions is referred to as molecular neuropharmacology, and it aims to discover medications that improve brain function [1].

Both domains deal with the interplay of neurotransmitters, neuropeptides, neuro hormones, neuromodulators, enzymes, second messengers, co-transporters, ion channels, and receptor proteins in the central and peripheral nervous systems. Researchers are creating medications to treat a variety of neurological problems, including pain, neurodegenerative diseases like Parkinson's disease and Alzheimer's disease, psychological disorders, addiction, and many more, by studying these interactions [2].

Neurochemical Interactions

Understanding how human behaviour and thought processes are passed from neuron to neuron, as well as how drugs might change the chemical foundations of these processes, is critical to understanding the potential advancements in medicine that neuropharmacology can provide. Neurons are called excitable cells because they have a lot of proteins called ion-channels on their surface membrane that allow small charged particles to move in and out [3].

Chemical information is received by the dendrites of the neuron, propagated through the perikaryon (cell body) and down the axon and eventually passed on to other neurons *via* the axon terminal. These voltage-gated ion channels allow the cell to quickly depolarize.

This depolarization will generate an action potential if it reaches a particular threshold. The influx of calcium ions into the cell occurs when the action potential reaches the axon terminal. Vesicles, little packets filled with neurotransmitters, attach to the cell membrane and release their contents into the synapse as a result of the calcium ions.

The pre-synaptic neuron interacts with the neurotransmitters released, while the post-synaptic neuron interacts with the neurotransmitters released. Once released into the synapse, the neurotransmitter can either attach to receptors on the post-synaptic cell, is re-up taken and saved for further transmission by the pre-synaptic cell, or be broken down by enzymes unique to that neurotransmitter in the synapse. These three effects are the three main ways in which drugs can impact cell transmission [4].

On a post-synaptic neuron, neurotransmitters interact with two types of receptors. LGICs, or Ligand-Gated Ion Channels, are the earliest type of receptor. LGIC receptors are the quickest types of chemical-to-electrical signal transducers. When a neurotransmitter attaches to a receptor, a conformational change occurs, allowing ions to flow directly into the cell. G-Protein-Coupled Receptors, or GPCRs, are the second category. Due to the increased number of metabolic events that must occur intracellular, these are much slower than LGICs. When a neurotransmitter attaches to a GPCR protein, it sets off a chain of intracellular events that can affect cellular biochemistry, physiology, and gene expression in a variety of ways. In the study of neuropharmacology, neurotransmitter/receptor interactions are crucial since many medicines currently being developed aim to disrupt this binding process [5].

Molecular Neuropharmacology

The purpose of molecular neuropharmacology is to discover new medications to treat neurological problems such as pain, neurodegenerative diseases, and psychological disorders by studying neurons and their neurochemical interactions, as well as receptors on neurons (also known in this case as neuro psychopharmacology)[6].

When discussing neurotransmission and receptor action, a few technical terms must be defined:

1. Agonist a chemical that binds to the protein of a receptor and activates it.

2. Competitive antagonist a chemical that binds to the same location on the receptor protein as the agonist, blocking the receptor from being activated.

3. Non-competitive antagonist a chemical that binds to a receptor protein at a different position than the agonist yet causes the protein to change shape, preventing activation.

Synthetic chemicals that behave as one of the three above can impact the following neurotransmitter/receptor interactions. Action potential inhibitory effects can also be induced by manipulating sodium/potassium ion channels throughout a cell [7].

Medication development will continue to improve as technology and our understanding of the nervous system grows, resulting in increased drug sensitivity and specificity. Structure activity interactions are a key topic of research in neuropharmacology, and they involve altering the chemical structures of bioactive chemical compounds to change their effect or potency (i.e., activity).

Central Nervous System

In the central nervous system, the GABA neurotransmitter mediates rapid synaptic inhibition. When GABA is produced from a pre-synaptic neuron, it binds to a receptor (most likely the GABAA receptor) in the post-synaptic cell, causing it to hyperpolarize (stay below its action potential threshold). This will neutralize the excitatory effects of other neurotransmitter/receptor interactions.

The key target for therapeutic research is the GABAA receptor, which has multiple binding sites that allow conformational alterations. Benzodiazepine, the most prevalent of these binding sites, has both agonist and antagonist effects on the receptor [8].

At this binding location, a popular medication called diazepam functions as an allosteric enhancer. A chemical called baclofen can enhance another GABA receptor known as GABAB. This chemical functions as an agonist, activating the receptor, and is known to aid in the control and reduction of spastic movement. The specific loss of dopaminergic neurons in the substantial naira characterizes Parkinson's disease, a neurodegenerative disease.

Levodopa, often known as L-DOPA, is the most commonly used medicine to treat this disease today [9]. Unlike the neurotransmitter dopamine, this precursor to dopamine can pass through the blood–brain barrier. There has been a lot of study done to see if L-dopa is a superior Parkinson's disease medication than other dopamine agonists. Long-term use of L-dopa, according to some, compromises neuro protection and, as a result, leads to dopaminergic cell death. Despite the lack of *in-vivo* or *in-vitro* evidence, some people still believe that long-term usage of dopamine agonists is better for the patient [10].

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